

TITLE PAGE

**Pendular Seesaw Nystagmus in a Patient with a Giant Pituitary Macroadenoma:
Pathophysiology and the Role of the Accessory Optic System
(Running Title: Pendular Seesaw Nystagmus Pathophysiology
and the Accessory Optic System)**

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Conflict of Interest Statement

No work resembling the enclosed article has been published or is being submitted for publication elsewhere. We certify that we have each made a substantial contribution so as to qualify for authorship and that we have approved the contents. We have disclosed that there is no financial support for this work or other potential conflicts of interests.

Keywords

Seesaw nystagmus; pituitary macroadenoma; accessory optic system, vestibulo-ocular reflex

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ABSTRACT

Seesaw nystagmus is characterized by cyclic eye movement with a conjugate torsional component and a dissociated vertical component. In the first half of the cycle, one eye elevates and intorts while the other eye depresses and extorts. The pattern is reversed in the remaining half of the cycle. We report a case of pendular seesaw nystagmus in a patient with a giant pituitary macroadenoma. Disturbance in the visuo-vestibular system is postulated to contribute to this form of seesaw nystagmus. Lesions compressing the optic chiasm and the accessory optic system could interrupt the transmission of retinal error signals to the inferior olivary nucleus and the interstitial nucleus of Cajal, thus interfering with the adaptive mechanism of the vestibulo-ocular reflex and leading to pendular seesaw nystagmus.

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11 **Pendular Seesaw Nystagmus in a Patient with a Giant Pituitary**
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17 **ABSTRACT**
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21 Seesaw nystagmus is characterized by cyclic eye movement with a conjugate
22 torsional component and a dissociated vertical component. In the first half of the
23 cycle, one eye elevates and intorts while the other eye depresses and extorts. The
24 pattern is reversed in the remaining half of the cycle. We report a case of pendular
25 seesaw nystagmus in a patient with a giant pituitary macroadenoma. Disturbance
26 in the visuo-vestibular system is postulated to contribute to this form of
27 nystagmus. Lesions compressing the optic chiasm and the accessory optic system
28 could interrupt the transmission of retinal error signals to the inferior olivary
29 nucleus and the interstitial nucleus of Cajal, thus interfering with the adaptive
30 mechanism of the vestibulo-ocular reflex and leading to pendular seesaw
31 nystagmus.
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46 ~~A 55-year-old female was admitted for progressive drowsiness and headache over~~
47 ~~one month. Three months before admission, she began to develop oscillopsia and~~
48 ~~blurring of vision. Neuro-ophthalmic examination showed reduced bilateral visual~~
49 ~~acuity to 6/60 (Snellen reading chart), bitemporal hemianopia and bilateral optic~~
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7 ~~atrophy. The most striking feature was the presence of pendular seesaw~~
8 ~~nystagmus. Magnetic resonance imaging revealed a pituitary mass with suprasellar~~
9 ~~extension compressing against the optic chiasm and ventral midbrain.~~

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14 ~~Disturbance in the visuo-vestibular system is postulated to contribute to seesaw~~
15 ~~nystagmus. Lesions compressing the optic chiasm and the accessory optic system~~
16 ~~could interrupt the transmission of retinal error signals to the inferior olivary~~
17 ~~nucleus, thus interfering with the adaptive mechanism of the vestibulo-ocular~~
18 ~~reflex and leading to pendular seesaw nystagmus.~~

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34 **Introduction**

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38 Seesaw nystagmus (SSN) is a rare ocular phenomenon characterized by the cyclic
39 movement of the eyes with two discrete elements, namely a conjunctive torsional
40 and dissociated vertical component. In the first half of the cycle, intorsion and
41 elevation are observed in one eye while extorsion and depression occurs in the
42 other. The movement pattern is reversed in the remaining half of the cycle.
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44 Nystagmography studies have classified this peculiar sign as exhibiting either a
45 pendular waveform or a jerky waveform (1-5). It is a rare ocular sign with only 50
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7 cases reported in the literature. We report a case of SSN in a patient with a giant
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9 pituitary macroadenoma and discuss the possible pathophysiology.

10 11 12 **Case Presentation**

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16 A 55-year-old Chinese female was admitted for progressive drowsiness and
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18 headache for one month. Three months prior to admission, she experienced
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20 oscillopsia and blurring of vision. On admission, the patient was ~~conscious and~~
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22 ~~alert and was eupituitarie. There was with~~ reduced bilateral visual acuity to 6/60
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24 with bitemporal hemianopia. Fundoscopy showed bilateral ~~band~~ optic atrophy
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26 ~~compatible with a chiasmatic or tract lesion~~. The most striking feature was the
27
28 presence of pendular-waveform seesaw nystagmus characterized by elevation with
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30 incyclotorsion in one eye and depression with excyclotorsion (counter-clockwise
31
32 torsion) of the fellow eye in all gaze positions (see video). The vertical and rotatory
33
34 oscillations continuously alternated with each eye in the absence of a fast phase.
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36 There was ~~no exacerbation of symptoms with head movement and there was~~
37
38 neither oculomotor nor abducens nerve palsy. ~~There were also no signs of~~
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40 ~~vestibular disturbance~~.

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43 Serum thyroid-stimulating hormone (TSH), free thyroxine (T4), morning cortisol
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45 and prolactin levels were all within normal limits. Magnetic resonance imaging
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47 (MRI) depicted a giant pituitary tumor (5.8cm (height) x 5.7cm 9 (width) x 6.3cm
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49 (length)) in size (Fig. 1). The lesion extended into the parasellar region and
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51 encased the cavernous segment of the right internal carotid artery (Knosp-Steiner
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7 grade IV). Superiorly, the lesion compressed against the optic chiasm and anterior
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9 third ventricle causing obstructive hydrocephalus. It also abutted the right meso-
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11 diencephalic junction.

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14 Craniotomy and near total excision of the pituitary tumor was performed via the
15
16 anterior interhemispheric transcallosal approach. The histopathological diagnosis
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18 was a non-functional pituitary adenoma. The patient's visual acuity did not show
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20 significant improvement and her bitemporal hemianopia as well as nystagmus
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22 persisted six-months after the procedure.
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38 **Discussion**

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42 The term SSN was first coined by Maddox in 1914 when he observed a patient
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44 with involuntary seesaw-like eye movements and bitemporal hemianopia ~~of~~
45
46 ~~unknown origin~~ (6). ~~Although the pathogenesis of SSN remains elusive,~~ Two
47
48 distinct forms of SSN exist, exhibiting either a jerky or pendular movement
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50 waveform. Jerky SSN, also known as hemi-SSN, consists of slow torsional phases
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52 in one direction (~~a~~ half-cycle) and quick phases in the opposite direction for
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7 the remaining half-cycle. In contrast, pPendular SSN ~~instead~~ describes a slow
8 smooth oscillating ocular rolling movement (7). Ascertaining the ~~It is believed that~~
9 ~~the precise~~ pattern of SSN ~~can may describe indicate~~ the location of the lesion and
10 offers insights on the complex interaction of the mechanisms involved in gaze
11 stability.
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18 Pendular SSN: ~~Such patients are~~ is typically observed in patients such as ours
19 diagnosed with tumors of the parasellar region with compression of the optic
20 chiasm (1, 8) ~~(8)~~. Other etiologies include chiasm trauma, congenital achiasma and
21 methotrexate-induced optic demyelination (4, 9-12). The resulting characteristic
22 visual signs of optic chiasm compression, i.e. bitemporal hemianopia, ~~are~~
23 therefore frequently ~~observed associated~~ with ~~pendular this form of~~ nystagmus (5,
24 13). ~~In one case a resolution of SSN occurred following the recovery of bitemporal~~
25 ~~hemianopia (8).~~
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36 Although the pathogenesis of pendular SSN remains elusive, Nakada et al
37 theorized that dysfunction of the visuovestibular mechanisms that control eye
38 movement may play a pivotal role (5). Physiologically the vestibulo-ocular reflex
39 (VOR) ~~has a rotational componentis elicited;~~ when the head rotates around any
40 cardinal axis (pitch, yaw or roll) with the purpose of stabilizing images on the
41 retina. Vestibular detection of a change in head position generates simultaneous
42 stimulatory and inhibitory signals to the extra-ocular muscles to induce reciprocal
43 ocular counter-rotation about the same axis ~~by ocular counter-rolling about the~~
44 ~~same axis.~~ Although ~~eliciting triggering~~ the reflex does not require visual input,
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7 subcortical visuovestibular adaptive mechanisms are thought to exist to minimize
8 an overshoot of ocular counter-rotation activity (5, 14, 15). One such subcortical
9 mechanism involves the accessory optic system (AOS) that detects optokinetic
10 stimuli through direction-selective retinal ganglion cells (15-17). This allows the
11 AOS, that does not serve formed vision, to sense frame shift changes which can
12 lead to corrective eye movements after signal processing through the cerebellum.
13
14 During head movement ~~it regulated by~~ retinal error (known as retinal slip) signals
15 are conveyed through the AOS through the optic chiasm to the inferior olivary
16 nucleus (ION) which projects climbing fibers to and subsequently the inhibitory
17 cerebellar/floccular Purkinje cells of the cerebellum/flocculonodular lobe (Figure 2)
18 (5, 14, 15). Purkinje cells have been shown to transmit inhibitory signals to the
19 vestibular nuclei and are critical in mediating the VOR adaptive properties of the
20 AOS (5, 15, 18).
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34 Nakada et al proposed that interruption of retinal error signals at the level of the
35 optic chiasm could lead to a disinhibited VOR, independent of head
36 movement, and may be the underlying mechanism for pendular SSN (5). The
37 torsional component of the nystagmus was hypothesized to be due to an absence
38 of visual information from the temporal visual fields due to chiasm compression
39 with subsequent disruption of eye movement calibrations in the roll plane (5). But
40 given the rarity of pendular SSN in patients with bitemporal hemianopia
41 secondary to pituitary tumors, other pathogenic mechanisms need to be considered.
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43 We propose a complimentary hypothesis that requires the additional interruption
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7 of internuclear signals of the AOS and the preservation of the interstitial nucleus
8 of Cajal (INC), with its efferent projections, in order for pendular SSN to occur.
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12 To elucidate the pathophysiology behind the dissociated vertical oscillations (in
13 the pitch plane) that are the hallmark of pendular SSN, further understanding of
14 AOS anatomy is required ~~We propose that~~(14, 17, 19). Three paired AOS terminal
15 midbrain nuclei receive visual signals from the contralateral retina through fibers
16 of the ~~transpeduncular optic tract (also known as the~~ accessory optic tract) that are
17 then relayed to the ipsilateral ION (14-16, 19). Rabbit electrical microstimulation
18 studies have revealed that each pair of AOS terminal nuclei processes visual
19 information regarding one of three cardinal rotational axes (14). In particular, the
20 medial-terminal ~~nucleus-nucleus (MTN)) of the AOS~~ processes retinal error
21 signals in the pitch plane (14, 15), ~~and~~ The MTN is anatomically located at the
22 ~~ventral midbrain tegmentum~~, bordered laterally by the cerebral peduncle and in
23 close proximity to the interpeduncular cistern (19) ~~and medially by the~~
24 ~~mammillary bodies. is particular~~ Of the three terminal nuclei of the AOS, the MTN
25 is the most vulnerable to anterior compression by ~~extremely~~ large suprasellar
26 lesions as suggested by our case ~~as in our case~~ (Figures 1 and 2). The
27 consequence of selective compression of the MTN could result in a lack of
28 transmission of retinal frame shift error signals in the pitch plane leading to
29 vertical nystagmus. The sparing of the more posteriorly located dorsal and lateral
30 terminal nuclei at the pretectal region may explain why pendular SSN lacks a
31 horizontal movement component.
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7 The INC, located at the dorsomedial midbrain tegmentum, is an important
8 constituent of the “eye movement neural integrator”, a distributed network of
9 neurons that combines ocular velocity signals and encodes them into position
10 commands (18). In particular, the INC is believed to be responsible for generating
11 vertical and torsional eye position signals and has extensive afferent and efferent
12 pathways to the vestibular nuclei that are conveyed via the medial longitudinal
13 fasciculus (7, 18, 20). Gamma aminobutyric acid (GABA) is a major inhibitory
14 neurotransmitter of the central nervous system and GABAergic neurons were
15 discovered to be predominantly active in governing AOS internuclear signaling
16 relative to excitatory stimulation (15). Injury to the MTN could lead to
17 disinhibition of the INC and several reports have emphasized the importance of its
18 preservation for pendular SSN to develop (4, 5, 7, 21). Stereotactic lesioning of
19 the INC in a patient was observed to abolish pendular SSN while electric
20 stimulation evoked its exacerbation (7). Our hypothesis is supported by our
21 patient’s MRI findings that confirm not only the integrity of the posteriorly located
22 INC, but also revealed considerable tumor compression of the optic chiasm and
23 the anterior interpeduncular region of the midbrain.

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42 Although an elucidation for the presence of the 180° out-of-phase dissociated
43 vertical eye movement observed in pendular SSN is lacking, a phylogenetic theory
44 has been suggested. Deregulation of the AOS with subsequent disinhibition of the
45 INC could represent an unmasking of an atavistic VOR pathway mediating
46 vertical eye movement observed in lateral-eyed animals (17). Lateral-eyed animals
47 (in contrast to frontal in humans) normally have dissociated binocular vision, i.e.
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7 when the head is tilted in the roll plane, one eye is turned superiorly and the other
8 eye is displaced inferiorly with corresponding torsional movement in order to keep
9 the eyes aligned along the horizon (17, 22). The emergence of pendular SSN could
10 reflect a regression to an older optokinetic system of our evolutionary
11 predecessors and corroborates current understanding of the neurologic mechanism
12 for infantile nystagmus (17).
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20 In contrast, jerky SSN is typically documented in patients with focal intrinsic
21 lesions of the midbrain involving the INC or its afferent central graviceptive
22 semicircular canal and/or otolithic projections from the vestibular nuclei (2). One
23 widely accepted theory is that this results in an imbalance in vestibular input from
24 the superior semicircular canals on both sides (3, 23, 24). Previously reported
25 lesions include infarction, cavernoma, hypothalamic hamartoma and multiple
26 sclerosis of the INC or along its afferent pathways in the mLF or medulla. (3, 25-
27 28). Unlike pendular SSN, visual impairment is often absent in these patients and
28 vestibular symptoms are more pronounced. Since the INC also has efferent
29 projections to the cervical spinal cord, lesions may also cause a contraversive
30 ocular tilt reaction, a type of postural synkinesis comprising of a head tilt to the
31 contralateral side, ipsilateral hypertropia and ocular counter-rolling (3, 23,
32 28, 29).
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48 In summary, both forms of SSN are related to the impairment of vestibular
49 responses initiated to maintain gaze stability during head rotation. Pendular SSN is
50 associated with miscalibration of retinal error signals (compromise of the
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7 visuovestibular adaptive mechanism) and jerky SSN results from an imbalance of
8 afferent vestibular signals to the INC (2). Their clinical presentations and
9 underlying causes are distinct (Table 1). This case supports the **concept** that
10 pendular SSN is a result of a double-hit involving a suprasellar lesion compressing
11 against the optic chiasm leading to bitemporal hemianopia as well as the MTN
12 leading to a disinhibited VOR.
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18 **FIGURE LEGEND**
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22 **Figure 1** Contrast T1-weighted MRI images showing an enhancing sellar mass
23 with suprasellar extension compressing against the optic chiasm superiorly and the
24 ventral midbrain (interpeduncular region) posteriorly (a: axial; b: sagittal; c:
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26 ventral coronal)
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31 **Figure 2** Proposed pathways involved in the pathophysiology of pendular SSN
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35 Double-hit hypothesis (compromise of the visuovestibular adaptive mechanism for
36 the VOR): First hit: Compression of the chiasm (CHM) causes loss of bilateral
37 temporal visual fields and retinal error miscalibration of the VOR leading to
38 nystagmus in the roll plane. Second hit: Compression of the medial terminal nucleus
39 (MTN) causes interruption of retinal error signals in the pitch plane: a) interruption of
40 inhibitory afferent GABAergic neurons to the the interstitial nucleus of Cajal (INC)
41 with subsequent vertical (pitch plane) and torsional (roll plane) nystagmus as
42 exhibited in previous INC stimulation studies; and b) disinhibition of the VOR due to
43 decreased cerebellar Purkinje fibers inhibitory signaling to the vestibular nuclei (VN).
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52 Abbreviations: AOS, accessory optic system; CHM, chiasm; ICN, interstitial nucleus
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7 of Cajal; ION, inferior olivary nucleus; MTN, medial terminal nucleus; VN,
8 vestibular nucleus; VOR, vestibulo-ocular reflex; III, oculomotor nerve nucleus, IV,
9 trochlear nerve nucleus; VI, abducens nerve nucleus (adapted from Simpson et al
10 (16)).
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18 **Figure 3.** Anatomical location of the AOS terminal nuclei in the midbrain at the level
19 of the superior colliculus
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23 The MTN is part of the AOS located at the ventral midbrain. A large suprasellar
24 tumor (e.g. a giant pituitary macroadenoma, dotted circle) may cause selective
25 compression of the MTN sparing more dorsally located nuclei such as the INC, lateral
26 terminal nuclei and dorsal terminal nuclei. The MTN conveys vertical plane retinal
27 error signals and interruption of this pathway may cause miscalibration of the VOR in
28 this plane. Abbreviations: AOS, accessory optic system; CN III, oculomotor nerve
29 nucleus; DTN; dorsal terminal nucleus; INC, interstitial nucleus of Cajal; LTN; lateral
30 terminal nucleus; MGB, medial geniculate body; mLF; medial longitudinal fasciculus;
31 MTN, medial terminal nucleus; NOT, nucleus of the optic tract (adapted from
32 Simpson et al (16)).
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5 **Pendular Seesaw Nystagmus in a Patient with a Giant Pituitary**
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7 **Macroadenoma:**
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9 **Pathophysiology and the Role of the Accessory Optic System**
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13 **ABSTRACT**
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18 Seesaw nystagmus is characterized by cyclic eye movement with a conjugate
19 torsional component and a dissociated vertical component. In the first half of the
20 cycle, one eye elevates and intorts while the other eye depresses and extorts. The
21 pattern is reversed in the remaining half of the cycle. We report a case of pendular
22 seesaw nystagmus in a patient with a giant pituitary macroadenoma. Disturbance
23 in the visuo-vestibular system is postulated to contribute to this form of
24 nystagmus. Lesions compressing the optic chiasm and the accessory optic system
25 could interrupt the transmission of retinal error signals to the inferior olivary
26 nucleus and the interstitial nucleus of Cajal, thus interfering with the adaptive
27 mechanism of the vestibulo-ocular reflex and leading to pendular seesaw
28 nystagmus.
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3 **Introduction**
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7 Seesaw nystagmus (SSN) is a rare ocular phenomenon characterized by the cyclic
8 movement of the eyes with two discrete elements, namely a conjunctive torsional
9 and dissociated vertical component. In the first half of the cycle, intorsion and
10 elevation are observed in one eye while extorsion and depression occurs in the
11 other. The movement pattern is reversed in the remaining half of the cycle.
12 Nystagmography studies have classified this peculiar sign as exhibiting either a
13 pendular waveform or a jerky waveform (1-5). It is a rare ocular sign with only 50
14 cases reported in the literature. We report a case of SSN in a patient with a giant
15 pituitary macroadenoma and discuss the possible pathophysiology.
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32 **Case Presentation**
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38 A 55-year-old Chinese female was admitted for progressive drowsiness and
39 headache for one month. Three months prior to admission, she experienced
40 oscillopsia and blurring of vision. On admission, the patient was alert with reduced
41 bilateral visual acuity to 6/60 with bitemporal hemianopia. Fundoscopy showed
42 bilateral band optic atrophy compatible with a chiasmatic or tract lesion. The most
43 striking feature was the presence of pendular-waveform seesaw nystagmus
44 characterized by elevation with incyclotorsion in one eye and depression with
45 excyclotorsion (counter-clockwise torsion) of the fellow eye in all gaze positions (see
46 video). The vertical and rotatory oscillations continuously alternated with each eye
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3 in the absence of a fast phase. There was neither oculomotor nor abducens nerve
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6 palsy. There were also no signs of vestibular disturbance.

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8 Serum thyroid-stimulating hormone (TSH), free thyroxine (T4), morning cortisol
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10 and prolactin levels were all within normal limits. Magnetic resonance imaging
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12 (MRI) depicted a giant pituitary tumor (5.8cm (height) x 5.7cm 9 (width) x 6.3cm
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14 (length)) in size (Fig. 1). The lesion extended into the parasellar region and
15
16 encased the cavernous segment of the right internal carotid artery (Knosp-Steiner
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18 grade IV). Superiorly, the lesion compressed against the optic chiasm and anterior
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20 third ventricle causing obstructive hydrocephalus. It also abutted the right meso-
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22 diencephalic junction.
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31 Craniotomy and near total excision of the pituitary tumor was performed via the
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33 anterior interhemispheric transcallosal approach. The histopathological diagnosis
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35 was a non-functional pituitary adenoma. The patient's visual acuity did not show
36
37 significant improvement and her bitemporal hemianopia as well as nystagmus
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39 persisted six-months after the procedure.
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Discussion

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5 The term SSN was first coined by Maddox in 1914 when he observed a patient
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7 with involuntary seesaw-like eye movements and bitemporal hemianopia (6). Two
8
9 distinct forms of SSN exist, exhibiting either a jerky or pendular movement
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11 waveform. Jerky SSN, also known as hemi-SSN, consists of slow torsional phases
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13 in one direction (a half-cycle) and quick phases in the opposite direction for the
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15 remaining half-cycle. In contrast, pendular SSN describes a slow smooth
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17 oscillating ocular rolling movement (7). Ascertaining the pattern of SSN may
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19 indicate the location of the lesion and offers insights on the complex interaction of
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21 the mechanisms involved in gaze stability.
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30 Pendular SSN is typically observed in patients such as ours diagnosed with tumors
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32 of the parasellar region with compression of the optic chiasm (1, 8). Other
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34 etiologies include chiasm trauma, congenital achiasma and methotrexate-induced
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36 optic demyelination (4, 9-12). The resulting characteristic visual signs of optic
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38 chiasm compression, i.e. bitemporal hemianopia, are therefore frequently
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40 associated with this form of nystagmus (5, 13).
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48 Although the pathogenesis of pendular SSN remains elusive, Nakada et al
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50 theorized that dysfunction of the visuovestibular mechanisms that control eye
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52 movement may play a pivotal role (5). Physiologically the vestibulo-ocular reflex
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54 (VOR) is elicited when the head rotates around a cardinal axis (pitch, yaw or roll)
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56 with the purpose of stabilizing images on the retina. Vestibular detection of a
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1 change in head position generates simultaneous stimulatory and inhibitory signals
2 to the extra-ocular muscles to induce reciprocal ocular counter-rotation about the
3 same axis. Although triggering the reflex does not require visual input, subcortical
4 visuovestibular adaptive mechanisms are thought to exist to minimize an
5 overshoot of ocular counter-rotation activity (5, 14, 15). One such subcortical
6 mechanism involves the accessory optic system (AOS) that detects optokinetic
7 stimuli through direction-selective retinal ganglion cells (15-17). This allows the
8 AOS, that does not serve formed vision, to sense frame shift changes which can
9 lead to corrective eye movements after signal processing through the cerebellum.
10 During head movement retinal error (known as retinal slip) signals are conveyed
11 through the AOS to the inferior olivary nucleus (ION) which projects climbing
12 fibers to the floccular Purkinje cells of the cerebellum (Figure 2) (5, 14, 15).
13 Purkinje cells have been shown to transmit inhibitory signals to the vestibular
14 nuclei and are critical in mediating the VOR adaptive properties of the AOS (5, 15,
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40 Nakada et al proposed that interruption of retinal error signals could lead to a
41 disinhibited VOR, independent of head movement, and may be the underlying
42 mechanism for pendular SSN (5). The torsional component of the nystagmus was
43 hypothesized to be due to an absence of visual information from the temporal
44 visual fields due to chiasm compression with subsequent disruption of eye
45 movement calibrations in the roll plane (5). But given the rarity of pendular SSN
46 in patients with bitemporal hemianopia secondary to pituitary tumors, other
47 pathogenic mechanisms need to be considered. We propose a complimentary
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1 hypothesis that requires the additional interruption of internuclear signals of the
2 AOS and the preservation of the interstitial nucleus of Cajal (INC), with its
3 efferent projections, in order for pendular SSN to occur.
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10 To elucidate the pathophysiology behind the dissociated vertical oscillations (in
11 the pitch plane) that are the hallmark of pendular SSN, further understanding of
12 AOS anatomy is required (14, 17, 19). Three paired AOS terminal midbrain nuclei
13 receive visual signals from the contralateral retina through fibers of the accessory
14 optic tract that are then relayed to the ipsilateral ION (14-16, 19). Rabbit electrical
15 microstimulation studies have revealed that each pair of AOS terminal nuclei
16 processes visual information regarding one of three cardinal rotational axes (14).
17 In particular, the medial terminal nucleus (MTN) processes retinal error signals in
18 the pitch plane (14, 15). The MTN is anatomically located at the ventral midbrain,
19 bordered laterally by the cerebral peduncle and in close proximity to the
20 interpeduncular cistern (19). Of the three terminal nuclei of the AOS, the MTN is
21 the most vulnerable to anterior compression by large suprasellar lesions as
22 suggested by our case (Figures 1 and 2). The consequence of selective
23 compression of the MTN could result in a lack of transmission of retinal frame
24 shift error signals in the pitch plane leading to vertical nystagmus. The sparing of
25 the more posteriorly located dorsal and lateral terminal nuclei at the pretectal
26 region may explain why pendular SSN lacks a horizontal movement component.
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56 The INC, located at the dorsomedial midbrain tegmentum, is an important
57 constituent of the “eye movement neural integrator”, a distributed network of
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1 neurons that combines ocular velocity signals and encodes them into position
2 commands (18). In particular, the INC is believed to be responsible for generating
3 vertical and torsional eye position signals and has extensive afferent and efferent
4 pathways to the vestibular nuclei that are conveyed via the medial longitudinal
5 fasciculus (7, 18, 20). Gamma aminobutyric acid (GABA) is a major inhibitory
6 neurotransmitter of the central nervous system and GABAergic neurons were
7 discovered to be predominantly active in governing AOS internuclear signaling
8 relative to excitatory stimulation (15). Injury to the MTN could lead to
9 disinhibition of the INC and several reports have emphasized the importance of its
10 preservation for pendular SSN to develop (4, 5, 7, 21). Stereotactic lesioning of
11 the INC in a patient was observed to abolish pendular SSN while electric
12 stimulation evoked its exacerbation (7). Our hypothesis is supported by our
13 patient's MRI findings that confirm not only the integrity of the posteriorly located
14 INC, but also revealed considerable tumor compression of the optic chiasm and
15 the anterior interpeduncular region of the midbrain.
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40 Although an elucidation for the presence of the 180° out-of-phase dissociated
41 vertical eye movement observed in pendular SSN is lacking, a phylogenetic theory
42 has been suggested. Deregulation of the AOS with subsequent disinhibition of the
43 INC could represent an unmasking of an atavistic VOR pathway mediating
44 vertical eye movement observed in lateral-eyed animals (17). Lateral-eyed animals
45 (in contrast to frontal in humans) normally have dissociated binocular vision, i.e.
46 when the head is tilted in the roll plane, one eye is turned superiorly and the other
47 eye is displaced inferiorly with corresponding torsional movement in order to keep
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1 the eyes aligned along the horizon (17, 22). The emergence of pendular SSN could
2 reflect a regression to an older optokinetic system of our evolutionary
3 predecessors and corroborates current understanding of the neurologic mechanism
4 for infantile nystagmus (17).
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12 In contrast, jerky SSN is typically documented in patients with focal intrinsic
13 lesions of the midbrain involving the INC or its afferent central graviceptive
14 semicircular canal and/or otolithic projections from the vestibular nuclei (2). One
15 widely accepted theory is that this results in an imbalance in vestibular input from
16 the superior semicircular canals on both sides (3, 23, 24). Previously reported
17 lesions include infarction, cavernoma, hypothalamic hamartoma and multiple
18 sclerosis of the INC or along its afferent pathways in the mLF or medulla. (3, 25-
19 28). Unlike pendular SSN, visual impairment is often absent in these patients and
20 vestibular symptoms are more pronounced. Since the INC also has efferent
21 projections to the cervical spinal cord, lesions may also cause a contraversive
22 ocular tilt reaction, a type of postural synkinesis comprising of a head tilt to the
23 contralateral side, ipsilateral hypertropia and ocular counter-rolling (3, 23, 28, 29).
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45 In summary, both forms of SSN are related to the impairment of vestibular
46 responses initiated to maintain gaze stability during head rotation. Pendular SSN is
47 associated with miscalibration of retinal error signals (compromise of the
48 visuovestibular adaptive mechanism) and jerky SSN results from an imbalance of
49 afferent vestibular signals to the INC (2). Their clinical presentations and
50 underlying causes are distinct (Table 1). This case supports the concept that
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pendular SSN is a result of a double-hit involving a suprasellar lesion compressing
against the optic chiasm leading to bitemporal hemianopia as well as the MTN
leading to a disinhibited VOR.

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3 **FIGURE LEGEND**
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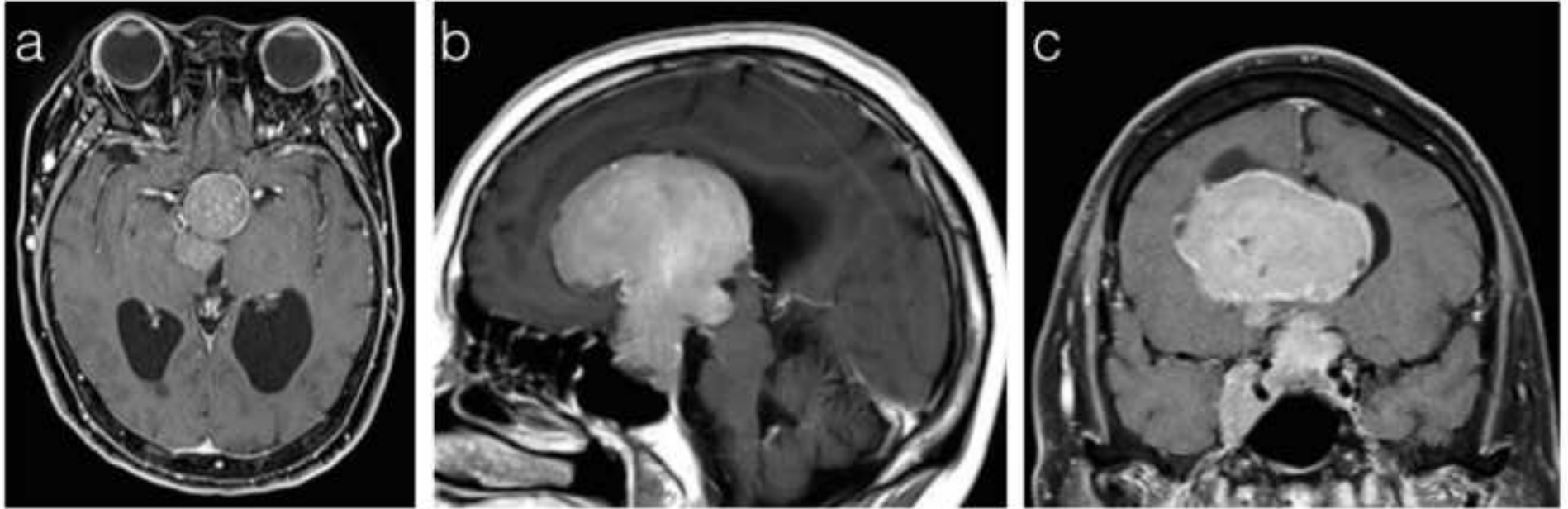
7 **Figure 1** Contrast T1-weighted MRI images showing an enhancing sellar mass
8 with suprasellar extension compressing against the optic chiasm superiorly and the
9 ventral midbrain (interpeduncular region) posteriorly (a: axial; b: sagittal; c:
10 coronal)
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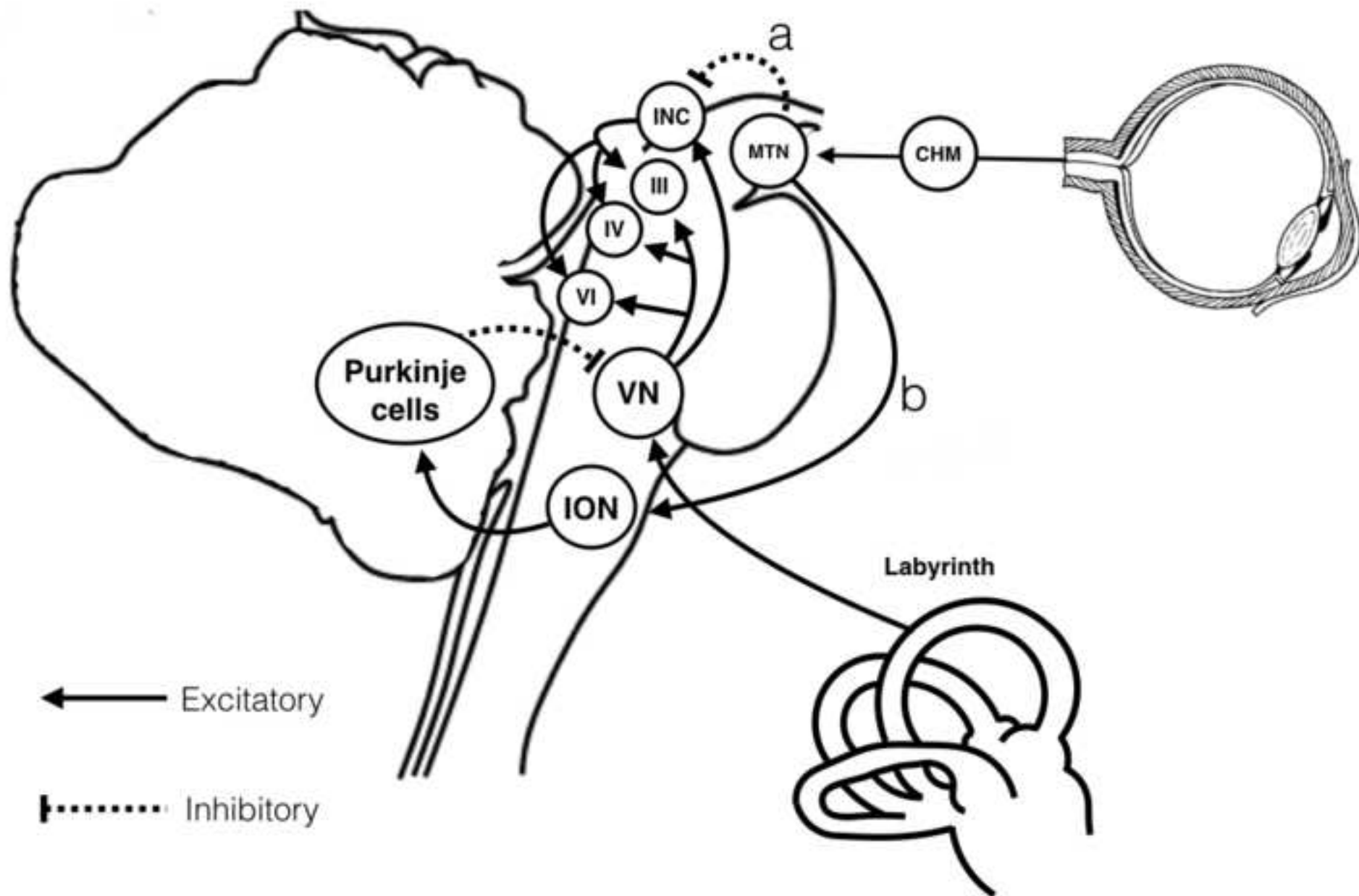
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19 **Figure 2** Proposed pathways involved in the pathophysiology of pendular SSN
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25 Double-hit hypothesis (compromise of the visuovestibular adaptive mechanism for
26 the VOR): First hit: Compression of the chiasm (CHM) causes loss of bilateral
27 temporal visual fields and retinal error miscalibration of the VOR leading to
28 nystagmus in the roll plane. Second hit: Compression of the medial terminal nucleus
29 (MTN) causes interruption of retinal error signals in the pitch plane: a) interruption of
30 inhibitory afferent GABAergic neurons to the the interstitial nucleus of Cajal (INC)
31 with subsequent vertical (pitch plane) and torsional (roll plane) nystagmus as
32 exhibited in previous INC stimulation studies; and b) disinhibition of the VOR due to
33 decreased cerebellar Purkinje fibers inhibitory signaling to the vestibular nuclei (VN).
34 Abbreviations: AOS, accessory optic system; CHM, chiasm; ICN, interstitial nucleus
35 of Cajal; ION, inferior olivary nucleus; MTN, medial terminal nucleus; VN,
36 vestibular nucleus; VOR, vestibulo-ocular reflex; III, oculomotor nerve nucleus, IV,
37 trochlear nerve nucleus; VI, abducens nerve nucleus (adapted from Simpson et al
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2 **Figure 3.** Anatomical location of the AOS terminal nuclei in the midbrain at the level
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4 of the superior colliculus
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9 The MTN is part of the AOS located at the ventral midbrain. A large suprasellar
10 tumor (e.g. a giant pituitary macroadenoma, dotted circle) may cause selective
11 compression of the MTN sparing more dorsally located nuclei such as the INC, lateral
12 terminal nuclei and dorsal terminal nuclei. The MTN conveys vertical plane retinal
13 error signals and interruption of this pathway may cause miscalibration of the VOR in
14 this plane. Abbreviations: AOS, accessory optic system; CN III, oculomotor nerve
15 nucleus; DTN; dorsal terminal nucleus; INC, interstitial nucleus of Cajal; LTN; lateral
16 terminal nucleus; MGB, medial geniculate body; mLF; medial longitudinal fasciculus;
17 MTN, medial terminal nucleus; NOT, nucleus of the optic tract (adapted from
18 Simpson et al (16)).
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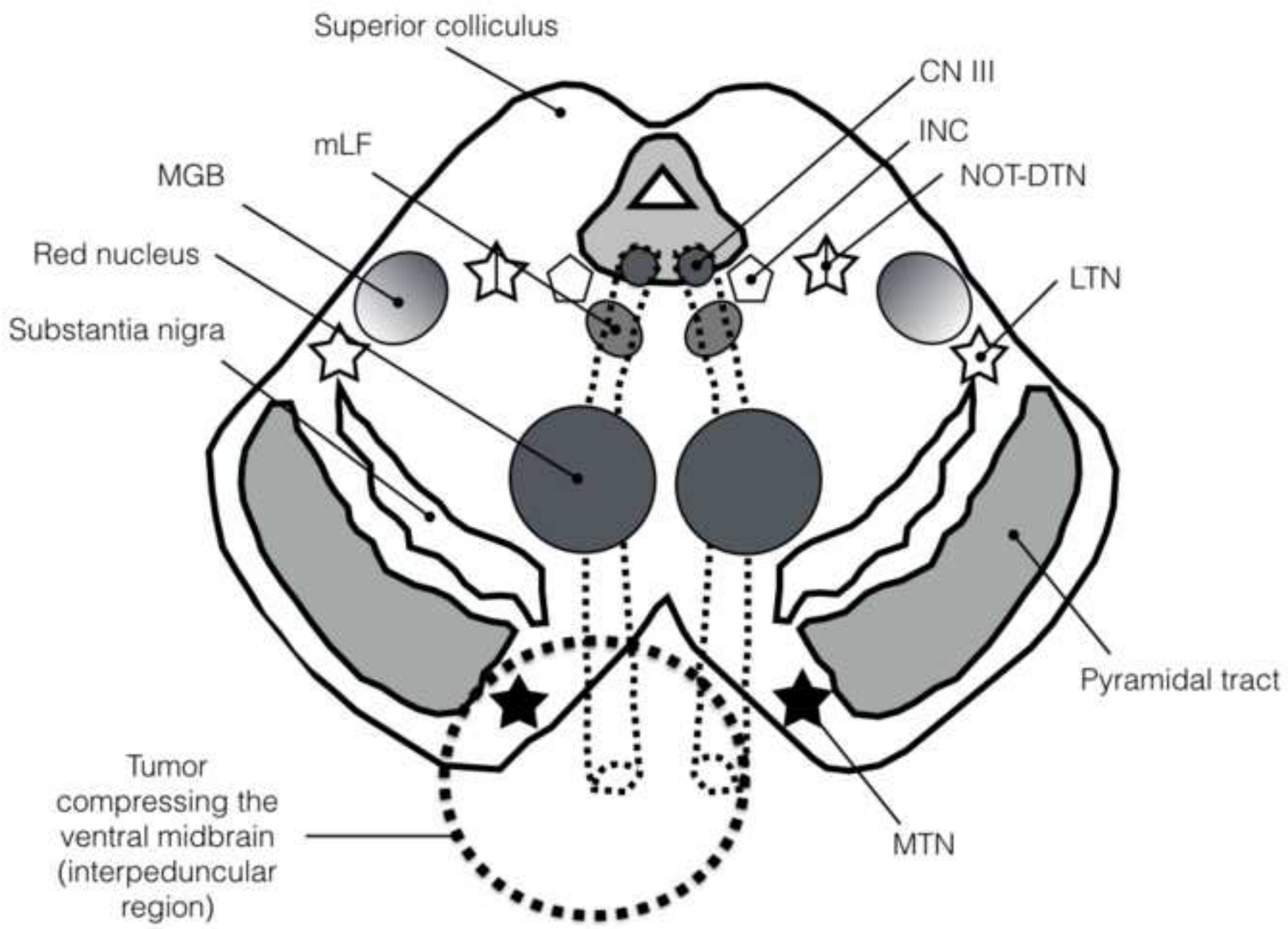


Table 1. Characteristics of the two forms of seesaw nystagmus

	Pendular SSN	Jerky SSN
Visual impairment	Yes	No
Nature nystagmus	SSN (torsion speed similar in each half cycle)	Hemi-SSN (fast and slow phases for each half-cycle)
Associated localizing signs	Bitemporal hemianopia (chiasm)	Ocular tilt reaction (INC) INO (mLF) Vestibular signs
Common etiology	Parasellar tumors	Infarct
Side of lesion	Midline	Unilateral (quick phase ipsilateral to lesion)

Abbreviations: SSN, seesaw nystagmus; INC, interstitial nucleus of Cajal; INO,

internuclear ophthalmoplegia; mLF, medial longitudinal fasciculus



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